THE RECIPROCAL EXCLUSION OF AMYLOIDOSIS--DISSEMINATED LUPUS ERYTHEMATOSUS

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and M. de Seze

Translation of: "L'exclusion reciproque amylose-lupus erythemateux dissemine," La Nouvelle Presse Medicale, Volume 3, Number 16, 1974, p. 1033

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16. Abstract

The authors have observed that within the realm of their clinical experience and in all reported cases save one in the literature the presence of LED mutually excludes amyloidosis and vice versa. In fact, the only known possible exceptions are cases of rheumatoid polyarthritis with amyloidosis and LE cells but without cutaneous or visceral manifestations of LED. The consensus of opinion is that these cases belong mainly to the clinical sphere of PR (and hence are susceptible to amyloidosis) and not LED.

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THE RECIPROCAL EXCLUSION OF AMYLOIDOSIS--DISSEMINATED LUPUS ERYTHEMATOSUS

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The Case of Polyarthritis with Lupiform Biology

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Amyloidosis can complicate chronic inflammatory illnesses, such as rheumatoid polyarthritis (P.R.). One also finds it in myelomas, notably those that are accompanied by Bence Jones proteinuria. This makes it even more surprising to ascertain its absence in disseminated lupus erythematosus (LED) that displays chronic inflammatory lesions, paints a clinical picture similar to PR and is accompanied by an elevation of serum levels and urinary excretion of short chain proteins [2]. If one considers that renal punch-biopsy is increasingly practiced for LED, it seems unlikely that the amyloid surplus could have been able to pass unnoticed in this affliction. Yet, we have never encountered it in 120 cases of LED seen at Viggo Petersen Center. We have collated 268 anatomical examinations of lupiform kidney in the literature. Amyloidosis is never mentioned. In his important series of 520 cases, Dubois [1] does not mention it. To our knowledge, a single case of LED, among thousands of published cases, that of Wegelius [5], describes the association of LED-amyloidosis.

On the other hand, we have observed recently [3] two cases (one personal, one of J. Ph. Mery in L. Morel-Maroger's histology text) of rheumatoid polyarthritis with lupiform biology without cutaneous or visceral signs of LED, complicated by amyloidosis. This association leans in favor of the widely adopted opinion that maintains that PR with lupiform biology (LE cells, antinuclear and anti-ADN antibodies) in the class of PR and not in that of LED.

The reciprocal exclusion of LED-amyloidosis has only been brought up a single time by Siguier et al. [4]. It raises however a thoroughly fascinating

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theoretical problem. It would be interesting to determine if NZB mice and their hybrids are capable, as are other strains of mice, of developing an experimental amyloidosis under the influence of various stimuli.

REFERENCES

1. Dubois, E. L., *Lupus erythematosus*, Vol. I, McGraw Hill edition, New York, 1966.
2. Epstein, W. V. and M. Tan, "Increase of L. Chain Proteins in the Sera of Patients with SLE and the Synovial Fluid of Patients with Peripheral R.A.," *Arthr. and Rheum.*, Vol. 9, p. 713, 1966.
3. Rousseau, J., *Reports on Two Cases of PR in Lupiform Biology which Developed Toward Amyloidosis*, Thesis, published in Paris (UER Broussais, Hotel Dieu), 1973.
4. Siguier, F., L. Orcel, P. Godeau and M. Dorra, "Amylosis and Collaginosis," *La maladie amyloide*, Volume 1, 296 pages, Massat edition, Paris, 1965.
5. Wegelius, O., "Amyloidosis of the Kidney, Adrenals and Spleen as a Complication of Acute SLE Treated with ACTH and Cortison," *Acta. Med. Scand.*, Volume 156, p. 91, 1956.

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